PREDICTION OF PROGNOSIS IN PATIENTS WITH CORONARY ARTERY DISEASE: POSSIBLE IMPROVEMENT OF PROGNOSIS WITH CORONARY ARTERY BYPASS GRAFTING

MEDICAL GRAND ROUNDS

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October 13, 1983

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Introduction

Determination of the prognosis of the patient with coronary artery disease is an important part of the patient's evaluation as this determination helps generate an overall perspective of the patient and formulate the plan of management. Furthermore, determination of the prognosis expected with both medical and surgical treatment is often the most important factor in the decision as to whether the patient should undergo coronary artery bypass grafting (CABG). Under certain circumstances, coronary angioplasty may be a more appropriate treatment. However, I will refer only to CABG with the assumption that angioplasty will be substituted for CABG, when appropriate.

As the relationship between prognosis and an increasing number of variables become known, the potential for accuracy in determination of prognosis increases. However, this increasing number of variables also increases the complexity and confusion of the process of determining prognosis. Much of this confusion is because a given patient may have apparently different prognoses when the prognoses are based on different patient variables. For example, several studies can be cited to show that a patient with 3 vessel coronary disease has a certain prognosis. In at least one randomized study, the prognosis of symptomatic patients with 3 vessel coronary disease is significantly better with CABG than under medical treatment. Some have taken this fact to mean that all patients with 3 vessel coronary disease should have surgery yet is this assumption correct? What if the patient with 3 vessel coronary disease is asymptomatic? There is evidence to suggest that overall, asymptomatic patients have a good prognosis. Therefore, judging the patient's prognosis from only the narrow viewpoints of either coronary anatomy or his symptomatic state may lead to different conclusions. In addition to the variables of coronary anatomy and symptomatic state, there are other patient variables which can be used to judge prognosis. The best patient evaluation should include as many of the important variables as possible.

Present evidence indicates that a patient should be characterized in terms of a minimum of four variables. These variables are listed in Table 1.

Table 1. Number of Subdivisions of Variables Important in Predicting Prognosis

Variable	Number of Subdivisions
Coronary Anatomy	4
Left Ventricular Function	4
Symptoms and/or Exercise Tolerance	3
Recent Infarction	2
Total Possible Combinations	96

Coronary anatomy should be subdivided as to whether 1, 2 or 3 vessel disease, or left main disease is present. Left ventricular function should be subdivided into

normal; minimal, moderate or severe dysfunction. Symptoms should be subdivided into asymptomatic, symptomatic, and unstable. Infarction should either be recent or not. These minimal subdivisions set up a matrix of 96 possible combinations. When viewed from the standpoint of determining a prognosis from a comprehensive understanding of the interplay of these variables, present knowledge is inadequate. There simply are not 96 or more adequate studies of each of these combinations. The problem is even more difficult when the question of the value of medical versus surgical therapy is raised. If each of these 96 combinations had a medical and surgical group for comparison, there would be almost 200 groups of patients. A reasonable estimate is that each group would need about 100 patients, consequently approximately 20,000 patients would have to be studied. Some have argued that the value of CABG needs to be studied by randomized trials. (1-9) Most would agree that this is ideal. However, when viewed from the perspective given above, trials such as would be needed would be very difficult to do, and from a practical standpoint, will never be done. For example, there are 4 randomized trials of CABG of over 200 patients which have either been completed or are underway. (10-13) These are listed in Table 2.

Table 2. Randomized Studies of Coronary Artery Bypass Grafting Consisting of Over 200 Patients

Study	Number of Patients
Completed	
VA Coop	686
European Coop	768
NHLI Unstable Angina	288
Underway	
CASS	780

These randomized trials have been conducted at a great deal of effort and expense. The information learned from these trials has been of great help, but the information is far short of that needed to answer the question of whether surgery should be done for the unique set of conditions of each individual patient. It is unlikely that larger trials will be conducted soon. Randomized trials of the treatment of coronary artery disease are particularly difficult because they must span a long period of time, frequently necessitating that new investigators pick up where the old investigators have left. (1) An additional problem that brings up ethical questions is that the purpose of these trials is to determine the value of medical versus surgical therapy in prolonging life. Surgery has already been shown to relieve angina. A frequent dilemma arises when a patient randomized to medical therapy has angina that could only be relieved by surgery. The investigator is then torn between his obligation to the patient and to science.

Because of these problems, a perfect guide to the determination of prognosis based on multiple patient variables is not now available, nor will be available soon. However, much information is available which can be used to construct a framework of the different variables within which the trend of the missing information can be seen more clearly.

In this discussion, I will give an overview of the relationship of the variables I have listed to prognosis. I will first treat each variable independently. Then, where possible, I will treat variables in conjunction with each other. Finally, I will compare each variable to its effect on the difference in prognosis of medical and surgical therapy.

Prognosis will be measured in terms of mortality. Although infarction is another method to judge prognosis, it is more difficult to define than death and therefore will not be used. In order to compare mortality rates from different studies, mortalities will usually be given in terms of annual mortality, which will simply be calculated by dividing the reported mortality by the years of followup. Although this method of comparison is not totally valid if mortality rates are not linear over time, the compromise will be made for ease of comparison. The mortality rates of patients not treated surgically will be termed medical annual mortality, while the mortality rates of patients after they have had surgery will be termed surgical annual mortality. Different forms of medical treatment will not be compared.

Several approaches will be used to help determine whether medical or surgical therapy affords the best prognosis. The simplest, and probably most often intuitively used, is the determination of the medical prognosis alone. If the medical annual mortality is low, then surgery can offer little or no improvement. On the other hand, if the medical annual mortality is high, then surgery potentially could help. A concept that has been suggested is that surgery tends to "fix" the prognosis at an annual mortality rate of 1-3%. (14-16) Therefore a surgical advantage or disadvantage can be implied based on whether the expected medical annual mortality is above or below this figure. This concept is, of course, highly speculative and unproven, but there is some evidence for it. The biggest group where it is of doubtful validity is the group of patients with poor left ventricular function. Medical and surgical therapy can be better compared when the mortality after each form of therapy is measured in a defined group of patients. The better defined and matched the groups are, the more valid is the comparison. (9) In some non randomized studies the matching of patients in the medical and surgical groups has been very thorough. (17-18) There is a tendency to accept these studies as being as valid as randomized studies. However, randomization not only is more effective in minimizing the difference between groups of measurable variables, but also of unmeasurable variables. (5) Two variables which are virtually never measured are the patient's overall condition and the integrity of the coronary vessels distal to the proximal coronary lesions. These factors are always subjectively assessed and enter into decision making to an unknown degree. Thus, when patients have had medical or surgical therapy following which results are retrospectively analyzed, an important question to ask is why were patients, who seemingly were identical, advised to have different forms of therapy.

Whether randomized or not, it must be remembered that even if medical and surgical treatment have the same effect on prognosis, there is a 1/20 chance that a difference in effect will be shown which is statistically significant at the .05 level. (19-20) This problem becomes much more serious when multiple subgroups are analyzed. For instance, if a study is done in which 10 subgroups are analyzed, and in reality there is no difference in treatment effect in any of the groups, there is still a 40% chance that one of the subgroups will show a statistically significant difference at the .05 level.

It is also important to remember that a study of the effect of two treatments has 3 outcomes. The study can show that the effects of the two treatments are statistically different; the study can show that the effects of the two treatments are statistically the same; or the study may neither show that the effects of treatment are different or the same. Frequently, it is assumed that a study which does not show a statistically significant difference of treatment automatically shows that the treatments are the same. But this is far from the case. In fact, in one study that looked at 71 "negative" randomized trials, the authors found that 67 of the 71 trials had a greater than 10% chance of missing a difference in treatment of 25%. (21)

With all of the problems in the studies and their designs, it is clear that a point will rarely be settled by one study. However, when a point is consistent throughout several studies, the point should be considered more valid.

Physiologic Relationship of Measured Variables to Death

The chief prognostic variables which are to be discussed subsequently are coronary anatomy, left ventricular function, symptomatic status, exercise test performance, and proximity to recent infarction. Before proceeding with the epidemiologic data, the physiologic rationale for these different variables will be put into perspective.

The sequence of events and their relationship to potential death in the patient with coronary disease is diagrammed in Figure 1.

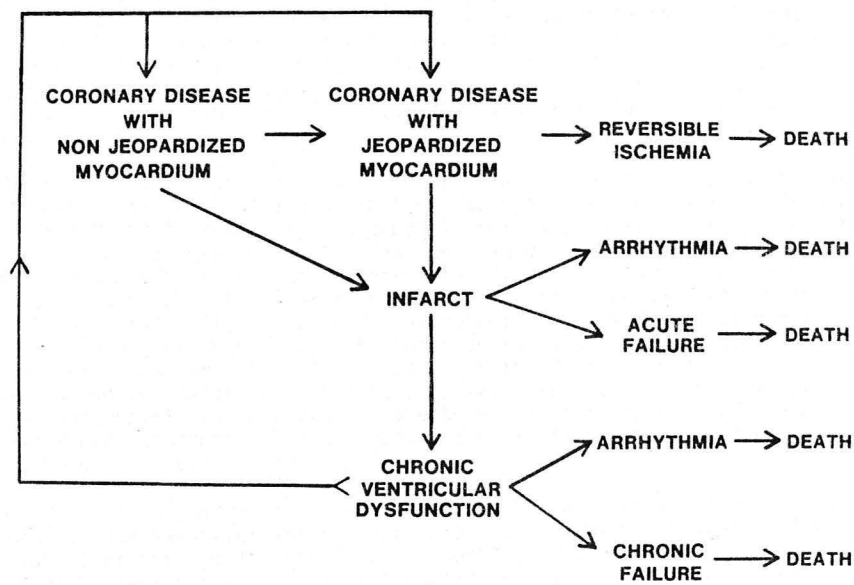

Figure 1. Physiologic progression of coronary artery disease with emphasis on the mechanism of death at different stages.

The coronary anatomy, as determined angiographically, is a measure of the extent of the basic pathologic problem. Anatomic coronary disease may result in no impairment to coronary flow; diminution in flow to a segment of myocardium sufficient to cause death of the segment; or a situation in which the flow is adequate to keep the muscle segment alive, but inadequate to supply the increased flow demands during times of myocardial stress. This latter condition in which flow is marginally adequate is said to result in "jeopardized myocardium." It may result from subtotal occlusion of a coronary vessel or collateral flow distal to a totally obstructed vessel. The presence of jeopardized myocardium can be inferred from the coronary anatomy as seen by angiography. However, it is probably better measured by stressing the ventricle with exercise and observing for evidence of ischemia by electrocardiography, symptomatology, radionuclide angiography, or perfusion scanning. The presence of jeopardized myocardium is best used as evidence of a tenuous supply-demand balance. The presence of jeopardized myocardium alone probably rarely causes death, as evidenced by the safety of routine exercise testing, when ischemia is regularly induced.

Outside of death, the most important event in the progression of coronary disease is infarction. If impending infarction could be predicted, the prognosis of a patient could be determined much more accurately than can be done at present. However, it is now known that infarction is usually caused by thrombosis in the coronary artery. (22-23) Although the thrombosis usually occurs in severely stenotic vessels, (24-26) there is not a good correlation between the severity of obstruction and occurence of thrombosis. Hence, measurement of the severity of myocardial jeopardy is imperfectly predictive of the probability of infarction.

Most of the deaths due to coronary disease occur soon after the onset of infarction. These are due either to fulminant hemodynamic failure or ventricular tachycardia-fibrillation. Other than acknowledging the high incidence of acute death with infarction, this phase will not be discussed further. If the patient does not die during the acute infarct period, his ventricle compensates to a variable degree. The left ventricular function with which the patient is left is dependent on the amount of myocardium infarcted and the extent of compensation. The left ventricular function with which the patient is left is another important prognostic factor.

Patients in the stage of chronic ventricular dysfunction may die because of chronic heart failure, ventricular tachycardia-fibrillation generated in the scarred myocardium, or by repeating the overall cycle and reinfarcting. The presence of chronic heart failure is usually easy to diagnose. However, the determination of whether a patient with chronic ventricular dysfunction dies because of a ventricular arrhythmia which is due to myocardial scarring or to reinfarction is more difficult to decide. It is apparent that both mechanisms cause death but their relative incidence is uncertain. The question is more than academic since CABG can potentially prevent future infarction, but is of little benefit in preventing arrhythmias due to myocardial scarring. (27-29) The approach to the patient with known serious ventricular arrhythmias due to myocardial scarring has been studied extensively and is generally considered more as an arrhythmia disorder than as a coronary artery disease disorder. (30-33)

Mortality is higher in the post infarction period than at other times. (34) Consequently, the variables which are helpful in determining prognosis during this period have been studied in much detail recently. The probable reason for the high mortality during the post infarct period is that a new coronary perfusion pattern and state of ventricular function is established. Patients that react

adversely to these new conditions are selected out and die.

Coronary Anatomy

The coronary anatomy as determined by coronary angiography, is a strong predictor of medical prognosis. Early studies done chiefly in the late 1960's first established this relationship and were summarized in a review in 1974. (35) Although the relationship between coronary anatomy and medical prognosis has been supported by other studies since this 1974 publication, the annual mortality for any given coronary anatomy has declined since that time. The results that will be presented are from these later studies. The reason for this apparent decrease in medical mortality is unknown. It may be secondary to an actual improvement in medical treatment, a difference in patient variables other than coronary anatomy, or to better angiographic resolution which would result in a given patient being graded as having worse anatomy.

The annual mortality of patients with single vessel disease in studies which look at just medical annual mortality, just surgical annual mortality, and both non randomized and randomized comparisons of medical and surgical mortality are shown in Table 3. It is apparent that in general the survival of patients characterized only as having single vessel disease is not increased by CABG.

These studies generally consist of patients with normal or only moderately abnormal ventricular function, and they represent a wide spectrum of clinical presentations. In the European Coop Study, all patients had an ejection fraction >.50 while in the VA Coop Study, 79% of the patients had an ejection fraction >.45. In the randomized studies, the investigator had to think it was ethically appropriate to either perform or withhold surgery on each patient. Therefore, patients with the least and most severe degrees of angina would tend to be eliminated.

Table 3. Medical and Surgical Annual Mortality of Patients with Single Vessel Disease

Center	Annual Mortality (%) Medical	Surgical	Followup (yrs)	Remarks
CASS (36)	2		4	
Baylor (37)		2	10	
Oregon (38)		1	10	Unstable Angina
Buffalo (39)		1	5	
Duke (40)	1.5	1.5	7	
VA Coop (10)	2	2	6	Randomized Stable Angina

The vessel which is diseased in single vessel disease may make some difference in prognosis. In Table 4 (40) the medical annual mortality of the different vessels is given. While these medical annual mortalities are not compared to surgery, it seems obvious that right coronary artery disease would benefit little from surgery.

Table 4. Medical Annual Mortality of Patients with Single Vessel Disease According to Disease Location

Vessel	Annual Mortality (%)	Followup (yrs)
Right	.5	7
Circumflex	2	7
Left Anterior Descending	2	7

If the disease is in the left anterior descending artery, prognosis varies depending on whether the lesion is proximal or distal. This is indicated in Table 5. The European Coop Study uses 2 vessel disease, but is included for corroborative evidence. The Duke Study is non randomized while the European Study is randomized.

Table 5. Medical Annual Mortality of Patients According to Whether LAD Disease is Proximal or Distal

Center	Disease	Annual Mortality % Medical	Surgical	Followup (yrs)
Duke (40)	Single LAD			
	Proximal	2		5
	Distal	.5		5
European Coop (11)	2 Vessel			
	Proximal LAD	3	1.5	6
	Distal LAD	1	1.5	6

Abbreviation: LAD = Left Anterior Descending Artery

Comparative studies of 2 vessel disease are shown in Table 6. Again little difference in annual mortality is apparent, chiefly due to the fact that the medical annual mortality is too low to be significantly improved upon.

Table 6. Medical and Surgical Annual Mortality of Patients with 2 Vessel Disease

Center	Annual Mortality (%) Medical	Surgical	Followup (yrs)	Remarks
CASS (36)	4		4	
Baylor (37)		3	10	
Oregon (38)		2	10	Unstable Angina
Buffalo (39)		1	5	
Duke (17)	2	1	7	Stable Angina Normal LV
European Coop (11)	2	2	6	Randomized Stable Angina Normal LV
VA Coop (10)	2	4	6	Randomized Stable Angina

Comparative studies of patients with 3 vessel disease are shown in Table 7. Medical annual mortality is higher with 3 vessel disease than with 1 or 2 vessel disease. However, surgical annual mortality remains low. Thus, a trend toward a beneficial effect of surgery is shown with 3 vessel disease. In the randomized European and VA Cooperative Studies, the beneficial effect of surgery is statistically significant. (In the VA Study, the difference in survival with 3 vessel disease is significant if the 10 hospitals with acceptable operative mortality are used and is not significant if all 13 participating hospitals are used).

Table 7. Medical and Surgical Annual Mortality of Patients with 3 Vessel Disease

Center	Annual Mortality (%) Medical	Surgical	Followup (yrs)	Remarks
CASS (36)	8		4	
Baylor (37)		5	10	
Oregon (38)		1.5	10	Unstable Angina
Buffalo (39)		1	5	
Duke (17)	2	1	7	Stable Angina Normal LV
European Coop (11)	3	1	6	Randomized Stable Angina Normal LV
VA Coop (10)	6	3	6	Randomized Stable Angina

Patients with left main disease have a poor prognosis when managed medically. The high medical annual mortality rate of these patients is shown in Table 8. Because of the low surgical annual mortality, there is a highly significant difference in prognosis between medical and surgical treatment.

Table 8. Medical and Surgical Annual Mortality of Patients with Left Main Disease

Center	Annual Mortality (%) Medical	Surgical	Followup (yrs)	Remarks
CASS (41)	10	3	3	
European Coop (11)	7	3	6	Randomized Stable Angina Normal LV
VA Coop (10)	10	3	3.5	Randomized Stable Angina

Both the severity of the stenosis of the left main artery and the presence of right coronary artery disease affect the prognosis of the patient with left main disease. It seems that these factors chiefly worsen medical annual mortality while causing little effect on surgical annual mortality. Thus, the worse the lesion and the presence of right coronary disease appear to increase the beneficial effects of surgery. These relationships are shown in Tables 9 and 10.

Table 9. Medical and Surgical Annual Mortality of Patients with Left Main Disease Subdivided According to Severity of Lesion

Center	Stenosis Severity (%)	Annual Mortality (%) Medical	Surgical	Followup (yrs)	Remarks
Duke (42)	50 - 70	11		3	
	> 70	19			
Montreal (43)	50 - 65	10	3	5	
	> 70	10	4		
CASS (41)	50 - 59	6	2	3	
	60 - 69	12	3		
	70 - 79	11	3		
	> 80	17	3		
VA Coop (15)	50 - 75	5	2	3.5	Randomized
	> 75	15	5		

Table 10. Medical and Surgical Annual Mortality of Patients with Left Main Disease Subdivided According to Presence of Right Coronary Disease

Center	Right Coronary Disease	Annual Mortality (%) Medical	Surgical	Followup (yrs)
Montreal (43)	Absent	6	2	5
	Present	12	5	
CASS (41)	Absent	4	2	3
	Present	12	3	

The overall relationship between coronary anatomy and prognosis can be seen more clearly when the information in the preceding tables is combined in Figure 2. The dotted lines connect the medical and surgical annual mortality data points when the data is from a randomized study.

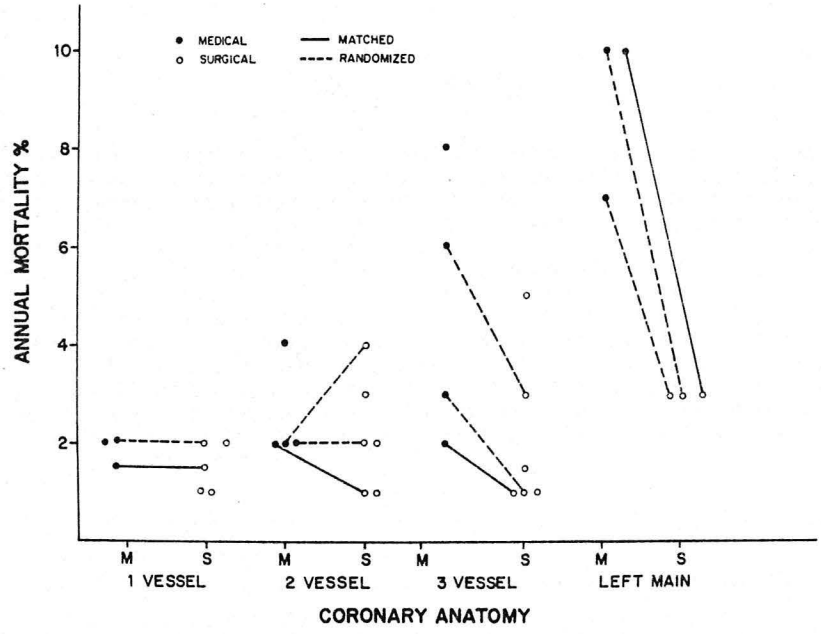

Figure 2. Medical and surgical annual mortality of patients subdivided according to coronary anatomy. The solid lines indicate a matched, but non randomized study. The dotted lines indicate a randomized study.

The graph illustrates the dependence of medical annual mortality on coronary anatomy. Furthermore, the graph illustrates the relative independence of surgical annual mortality on coronary anatomy. Points connected by a dotted line indicate a randomized study. Thus, the advantage of surgery in improving survival increases as the coronary disease becomes more severe. This graph is an illustration of the previously mentioned concept that the advantage of surgery depends on the medical prognosis.

In patients who are symptomatic, have good or moderately diminished left ventricular function, and have distal coronary arteries suitable for grafting:

Medical prognosis worsens as the anatomic extent of coronary disease becomes more severe

Surgical prognosis changes little as the anatomic extent of coronary disease becomes more severe

Surgical prognosis is better than medical prognosis in the presence of 3 vessel and left main coronary disease

Ventricular Function

Left ventricular function has long been recognized as an important predictor of prognosis. When not qualified further, the term 'ventricular function' in reference to coronary artery disease is usually meant to imply the function of the left ventricle in the resting or non stressed state. It is usually measured at cardiac catheterization or by radionuclide angiography. Results are usually expressed as the ejection fraction. The normal average ejection fraction is .67 and the lower limit of normal is .50. Ventricular function and the severity of coronary disease are not well correlated, (44) consequently they are independent predictors of prognosis. In Table 11, the medical annual mortality of patients is shown. The patients are grouped by number of vessels diseased and ventricular function. These patients are from Duke (45) and the CASS Study (36), and were followed for 5 and 4 years respectively. The patients represent a wide spectrum of symptoms. The independent and cumulative effect of the severity of coronary disease and ventricular function is apparent.

Table 11. Medical Annual Mortality of Patients Subdivided According to Coronary Anatomy and Left Ventricular Function

Number of Diseased Vessels	CASS (36) Ejection Fraction			Duke (45) Ventricular Function		
	.50-1.00	.35-.49	≤ .34	Normal	Abnormal	Very Abnormal
1	1	2	6	1	4	
2	2	4	11	2	3	16
3	4	7	12	3	7	12

Soon after coronary bypass surgery became established, two important facts became apparent. The first fact was that surgery did not cause an improvement in ventricular function. The second fact was that operative mortality was highly dependent on ventricular function. In fact, as shown in Table 12, operative mortality was so high in patients with poor ventricular function that surgery was generally not considered for these patients.

Table 12. Operative Mortality for CABG Performed in About 1970

Center / Ejection Fraction	Operative Mortality %
Seattle (46)	
< .33	33
> .33	3
Boston (47)	
< .50	35
> .50	3
Pennsylvania (48)	
.12 - .30	25
.31 - .40	12
.41 - .50	4
> .50	4
Duke (49)	
< .25	55
> .25	4

However in the 1970's surgical techniques were improved and operative mortality in patients with poor ventricular function decreased dramatically. This later operative mortality, in comparison to ventricular function, is shown in Table 13. This table comes from the CASS Study which is a prospective study of over 6,000 patients from 15 centers (50), consequently, it is broadly representative. The patients were entered between 1974 and 1979.

Table 13. Operative Mortality for CABG Performed In 1974-79. (CASS Study)

Ejection Fraction	Operative Mortality %
< .19	7
.20 - .29	4
.30 - .39	2
.40 - .49	3
> .50	2

Thus, unreasonably high operative mortality is no longer an absolute reason to not operate on patients with poor ventricular function.

The annual mortality of patients with 1 vessel coronary disease, subdivided by ventricular function is shown in Figure 3.

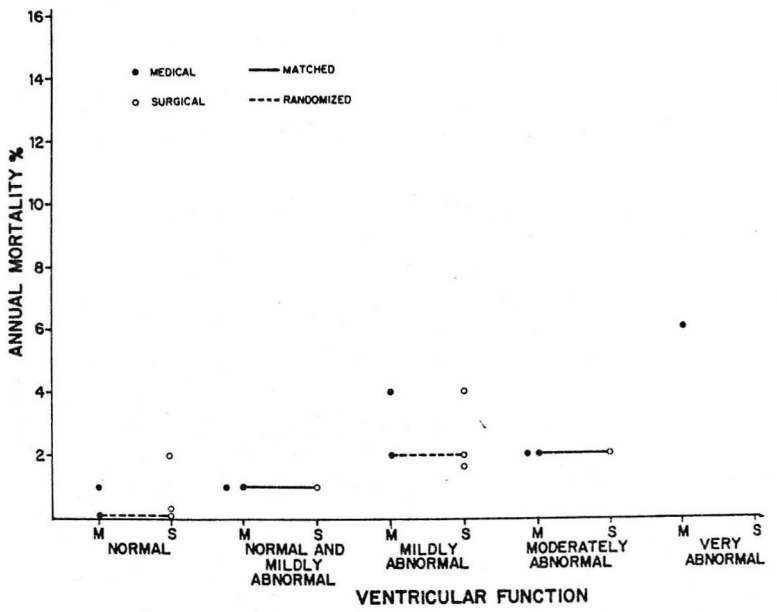

Figure 3. Medical and surgical annual mortality of patients with single vessel disease subdivided according to ventricular function.

This graph and the subsequent graphs in this section are based on studies which were reported in the late 1970's and early 1980's, consequently, they represent patients studied in the mid to late 1970's. (18,37,38,41,43,51,36,45) The patients represent a wide spectrum of symptoms. They have been followed for 4 to 10 years. Two points connected by a solid line means that the patients came from the same study while two points connected by a dotted line means that the patients were randomized. Because different methods of measuring ventricular function were used, the ventricular function groups used are somewhat arbitrary so that all studies could be shown together. Normal ventricular function means no wall motion abnormality whatsoever. Mildly abnormal function means that a wall motion abnormality is present, but that overall ventricular function is good with an ejection fraction

above .45 or .50. Moderately abnormal ventricular function means an ejection fraction from .30 - .35 to .45 - .50. Very abnormal ventricular function means an ejection fraction below .25 - .30. Notice that there is only a possible slight rise in medical and surgical annual mortality as ventricular function worsens. There is no difference in medical and surgical prognosis.

The annual mortality of patients with 2 vessel disease is shown in Figure 4.

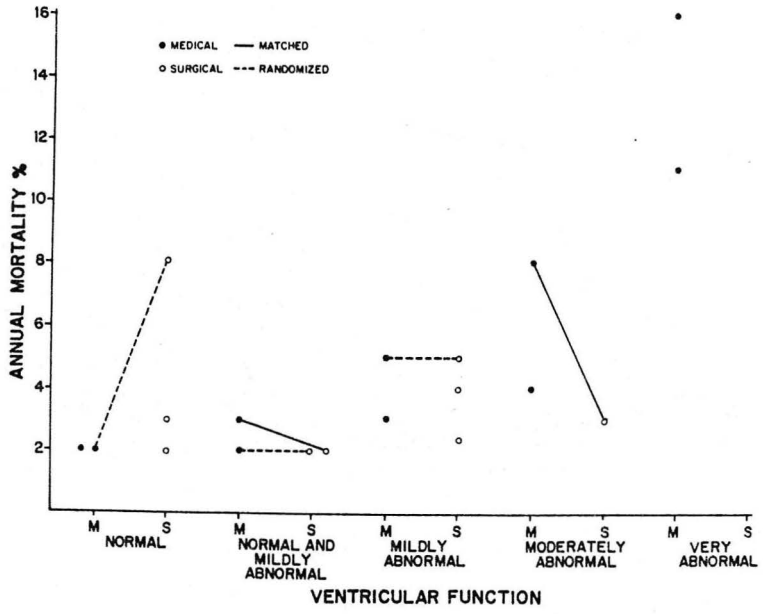

Figure 4. Medical and surgical annual mortality of patients with 2 vessel disease subdivided according to ventricular function.

Notice that the medical annual mortality is clearly dependent on ventricular function. There is insufficient information to make a statement about surgical annual mortality.

The annual mortality of patients with 3 vessel disease is shown in Figure 5.

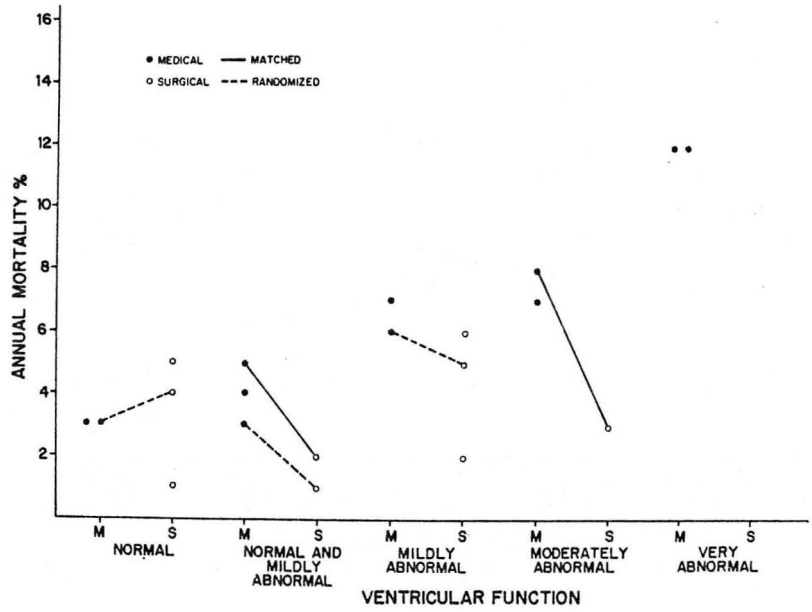

Figure 5. Medical and surgical annual mortality of patients with 3 vessel disease subdivided according to ventricular function.

Again notice that in patients with 3 vessel disease, the medical annual mortality increases with worsening ventricular function. The surgical annual mortality appears to improve over medical annual mortality as ventricular function worsens, but the data are insufficient to be conclusive.

The annual mortality of patients with left main disease is shown in Figure 6.

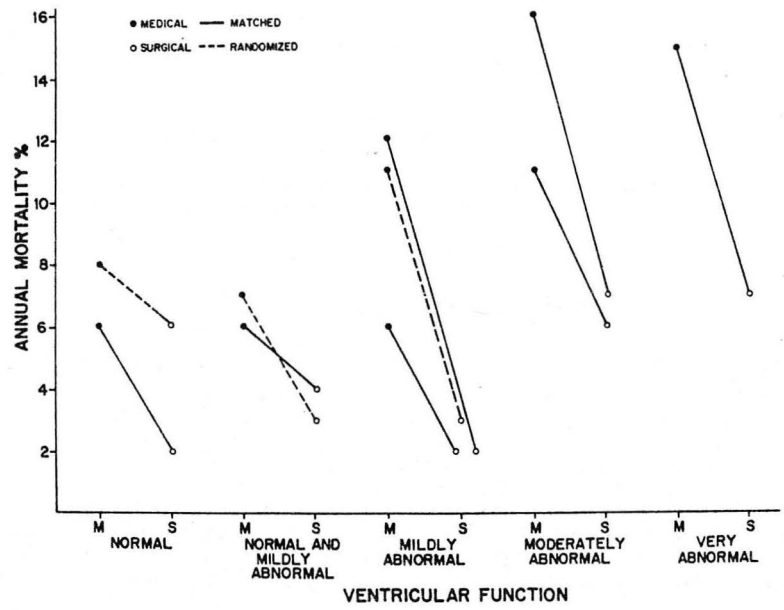

Figure 6. Medical and surgical annual mortality of patients with left main disease subdivided according to ventricular function.

On this graph, the high mortality of left main disease accentuates the correlation of mortality with ventricular function. Both medical and surgical annual mortality increase as ventricular function worsens. However, medical annual mortality increases more than surgical annual mortality with the result that surgery offers a better prognosis than medical treatment. This surgical advantage appears to increase as ventricular function worsens. An important reservation to this statement is that the studies in the two worse ventricular function groups were not randomized studies.

Both medical and surgical annual mortality increase as ventricular function worsens

Ventricular function contributes to annual mortality independently of coronary anatomy

Studies comparing medical and surgical annual mortality in the worst ventricular function groups are not randomized, however:

Worsening ventricular function seems to contribute independently to the improvement in prognosis caused by surgery

Symptoms and Exercise Tests

The symptomatic status and exercise test performance will be considered together because they are generally considered to be indicators of the degree to which myocardium is in jeopardy. Occasionally, the symptoms and exercise test performance will not agree as to the degree to which they indicate that myocardium is in jeopardy. An example is the asymptomatic patient with ST depression during an exercise test. The resolution of problems such as this can be complicated, however a simplistic solution is to try to decide what the combination of results best suggests in terms of myocardial jeopardy. If the answer is not clear, then an additional study such as coronary angiography or a different form of stress testing, such as radionuclide angiography or thallium scintigraphy, should be done.

Unstable Angina

Unstable angina is a syndrome generally defined as angina at rest or a worsening angina pattern. In the studies to be discussed, it has generally been a requirement that the syndrome be severe enough to warrant admission to a coronary care unit. Unstable angina has generally been thought to portend a worse prognosis than stable angina. However, the prognosis actually found in different studies varies widely. In this discussion, I will summarize only some of the most recent studies in which the patients were either well characterized or were randomized to medical or surgical therapy. The study of unstable angina is somewhat different from the study of other classification characteristics because patients usually have unstable angina only for a short time. Consequently, the usual study involves patients with angina who have a history of unstable angina.

Patients treated medically for unstable angina have a surprisingly low hospital mortality ranging from 0 - 5%. (12,52,53,54,55,56) In most studies, this death rate is not high enough to divide the prognosis on the basis of coronary anatomy. However, in one study of 188 patients with unstable angina, the hospital mortality was 4% overall. When subdivided by coronary anatomy, the hospital mortality was 30% in the 11% of patients with left main disease and 0.7% in the rest of the patients without left main disease. (55)

The medical annual mortality of patients in the 2-4 years after a period of unstable angina is 4-6%, (52,12,54,57) as shown in Figure 7.

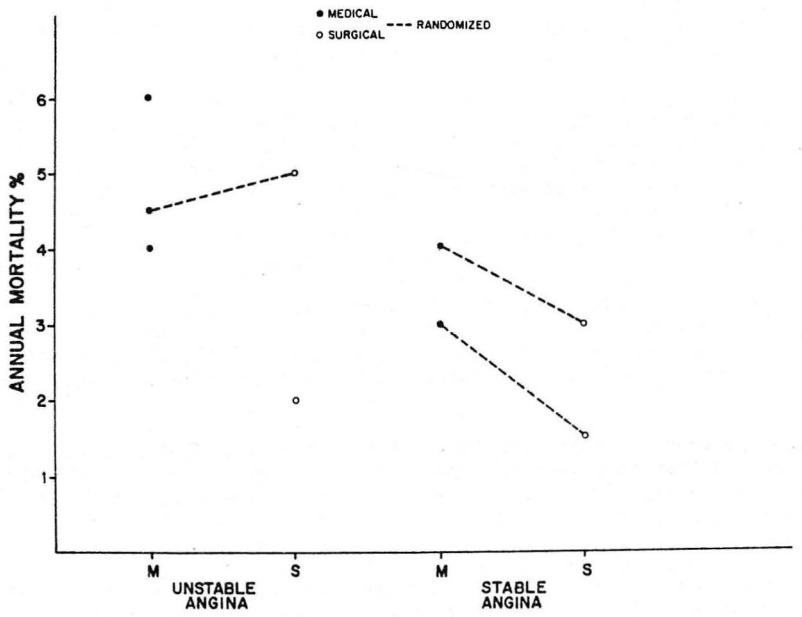

Figure 7. Medical and surgical annual mortality of patients with unstable angina. For comparison, the results of the VA and European Cooperative Study on stable angina are shown.

Mortality was not determined based on coronary anatomy, however there were approximately equal numbers of patients with 1, 2, and 3 vessel disease. Patients with left main disease were excluded from the studies with the 4 and 4.5% annual mortality. Patients with left main disease comprised 7% of the patients in the study with a 6% annual mortality. (57) As shown on the graph for comparison, the

medical annual mortality of the patients with unstable angina is similar to the medical annual mortality of the patients with stable angina in the VA and European Cooperative Studies. (10,11)

The operative mortality of patients with unstable angina varies from 1.8 - 8%. (58,12,50,38) As with stable angina, this mortality is dependent on the presence of left main disease and left ventricular function. (58) The presence of unstable angina itself probably increases the operative mortality only slightly. In the large CASS Study, the operative mortality was 3.5% for patients with unstable angina and 2.5% for patients with stable angina. (50)

The surgical annual mortality varies from 2% in a large study in which patients were followed 10 years postoperatively to 5% in the NHLI Study in which patients were followed for 2 years. (12) In the study with 10 year followup, there was no difference in surgical annual mortality between patients with 1, 2, and 3 vessel disease. (38) These surgical annual mortalities are also shown on the above graph along with the surgical annual mortalities of patients with stable angina in the VA and European Cooperative Studies. Here again the similarities of annual mortalities between patients with unstable and stable angina are apparent.

The 3 randomized studies of patients with unstable angina in which patients were followed for a mean time of 4, 18 and 30 months have shown no difference in medical or surgical therapy. (13,54,53) Only 1 of these studies is shown on the above graph because the mortality figures in the other studies could not be converted to annual mortality.

Several important points should be made about the foregoing results and discussion. The first point is that the studies were generally of patients who presented with unstable angina who had not received intensive medical therapy. If the patients promptly responded to medical therapy and stabilized, they were still considered to be in the unstable group. Consequently, the patients in these studies may not be completely representative of the problem patients who are poorly responsive to medical therapy. The overall effect of this situation is that medical survival in these studies may be better than with the problem patient with unstable angina. The second point is that in the randomized studies, catheterization and surgery were generally performed soon after admission, before medical stabilization was obtained. It is now generally agreed that catheterization and surgery performed before medical stabilization entails more risk than after stabilization, and furthermore the delay while attempting stabilization does not materially increase the risk. (59,60,61,62,63) Therefore, the operative mortality and consequent surgical annual mortality in patients who have been stabilized may be better in current practice than indicated by these randomized studies. Finally, patients who are treated medically for unstable angina usually stabilize, but still have severe stable angina. Thus, even in the 3 randomized studies where an attempt was made to keep patients in their original treatment group, the crossover rate of patients from medical to surgical treatment was 36-42%. (12,54,53)

The medical and surgical annual mortalities of patients with unstable angina are probably little different than patients with stable angina

After stabilization of unstable angina, there is a high probability of severe stable angina.

Surgery should not be performed merely because of a history of unstable angina.

A suggested approach is to stabilize patients medically, then to reevaluate and base therapy on current clinical status, coronary anatomy, and ventricular function

Randomized studies have not shown a difference in mortality between medical and surgical therapy

These conclusions and recommendations apply to patients who have either not received maximal medical therapy or have responded to medicine. They do not apply to patients who are refractory to maximum medical therapy. In these patients surgery is generally indicated, but there are no studies of this situation

Asymptomatic State

The presence of coronary disease is usually detected in the asymptomatic patient either by the presence of a previous myocardial infarction or by coronary angiography after the finding of an abnormal electrocardiographic response to a screening exercise test. It is important to realize that coronary disease is present in only 26-37% of asymptomatic patients who have ST depression on exercise testing. (64) It is also important to realize that most articles with a title that suggests a study of asymptomatic patients include a substantial number of patients with previous infarctions. (65,72,66,67,68,79,18)

The only study in which a larger number of asymptomatic patients with proven coronary disease, who had not sustained a previous infarction, comes from Bs Air Force Base. Ninety patients were followed for an average of 57 months. Of these patients, only 2 died for an annual mortality of 0.5%. (69,70,71) Of these 90 patients, 22 developed cardiac events, of which 16 were new angina, 4 were infarction, and 2 sudden death.

The rest of the information comes from studies in which patients with previous myocardial infarctions were included. These patients were either asymptomatic or mildly symptomatic, and had been followed for 2-7 years. In Figure 8, the medical annual mortality of asymptomatic patients is shown, and where possible, is compared to the medical annual mortality of the symptomatic patients from the same institution. (65,72,18) These comparisons are indicated by the solid lines. This graph suggests that the annual mortality of asymptomatic patients is lower than symptomatic patients, but is still appreciable.

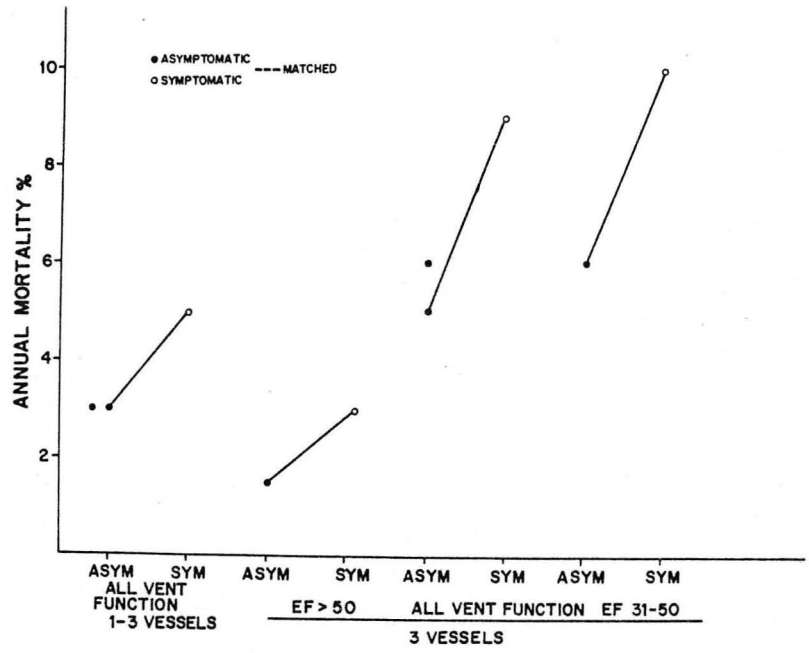

Figure 8. Annual mortality of asymptomatic and symptomatic patients subdivided according to ventricular function and coronary anatomy. The solid lines indicate the patients came from the same institution.

In the Figure 9, the results of several studies of the medical and surgical prognosis of asymptomatic or mildly symptomatic patients is shown. (66,67,68,18) In the results, the matched studies are connected by the solid lines. In the same study which shows a difference in mortality of 3 vessel disease with an ejection fraction of 31-50, patients with 1 and 2 vessel disease, both with ejection fractions of 31-50 and over 50, showed no difference in medical and surgical annual mortality. (18)

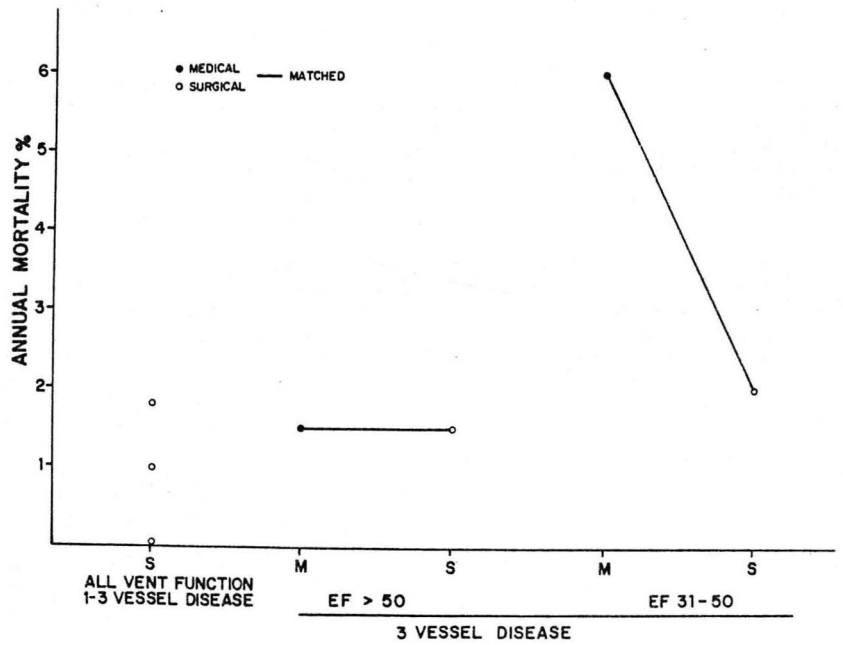

Figure 9. Medical and surgical annual mortality of asymptomatic or mildly symptomatic patients subdivided according to ventricular function and coronary anatomy.

Therefore, there is not much information on the asymptomatic patient. The available evidence suggests that asymptomatic patients with 3 vessel disease and diminished ventricular function may have an improved prognosis with surgery. However, patients with 1 and 2 vessel disease, and those with 3 vessel disease and good ventricular function do not have an improved prognosis with surgery. In general, this question of the asymptomatic patient should be approached in conjunction with the exercise test. More information on the minimally symptomatic patient may be available soon since it is one of the goals of the randomized portion of the CASS study now underway. (13)

Exercise Test

Clinical exercise testing (73) usually calls for the patient to exercise on a bicycle or treadmill as the resistance to pedaling, speed, or grade is gradually increased. At some point, the patient will have to stop because of the onset of some symptom. If the patient is limited because of coronary artery disease, this symptom will usually be secondary to stress induced ischemia of the ventricle. Thus, the symptom will be angina, due to the ischemia; or dyspnea, fatigue, or dizziness, due to ventricular dysfunction secondary to the ischemia. Therefore, the amount of exercise the patient can tolerate before any of these symptoms develop is an important measure of the functional reserve of the ventricle. The implication is that this exercise tolerance is inversely proportional to the degree to which myocardium is in jeopardy. A difficulty with the evaluation of exercise tolerance is that it is sometimes difficult to distinguish whether vaguely defined symptoms are secondary to ventricular ischemia or another etiology.

In studies of patients with coronary artery disease who were followed for 2-5 years after the exercise test, their medical annual mortality was dependent on the stage of the exercise test which they could enter. (i.e. Stage II means completion of Stage I). The results of these studies are shown in Figure 10. (74,75,76,77,78) Stage I indicates severe limitation while Stage IV is approximately a normal exercise tolerance. (73)

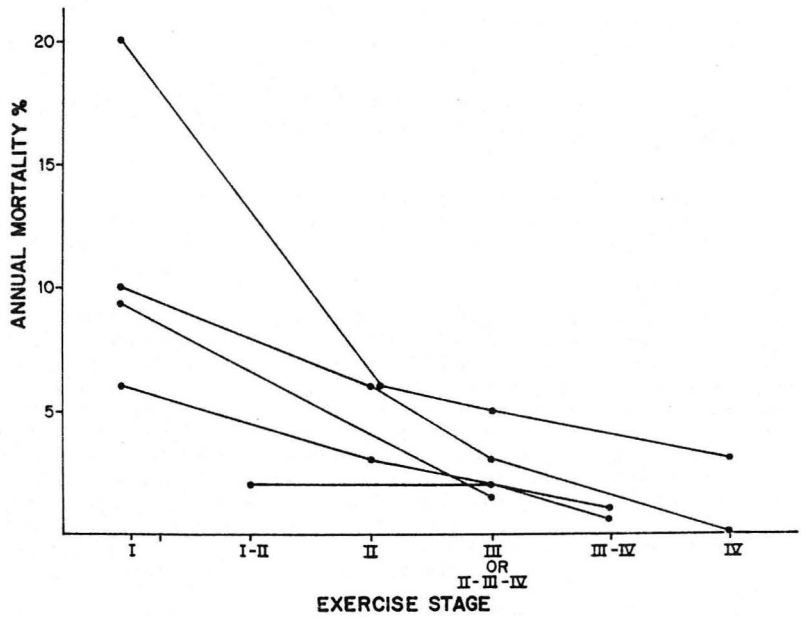

Figure 10. Medical annual mortality of patients subdivided according to stage of Bruce protocol that patient entered (i.e. Stage II means patient finished Stage I).

When the patient population is subdivided into groups based on both exercise tolerance and coronary anatomy, it is apparent that prognosis is independently correlated to both methods of classification. The medical annual mortality of patients subdivided in this manner are shown in Table 14. Unfortunately, information is not available for left main disease.

Table 14. Medical Annual Mortality of Patients Subdivided According to Exercise Tolerance and Coronary Anatomy

Center	Exercise Tolerance	Number of Diseased Vessels		
		1	2	3
Quebec (77)	I	0	5	14
	II	1	4	7
	III	2	3	3
NHLI (72)	poor	<2		9
	good			4

Abbreviations: I, II, III = Stages of Bruce protocol; poor = < 100 watts (approximately midway between Stage I-II); good = > 100 watts

From these studies, it appears that patients with a good exercise tolerance have a low medical annual mortality regardless of their coronary anatomy. However, patients with a poor exercise tolerance have widely divergent annual mortality depending on their coronary anatomy.

Another measurement usually made during exercise testing is the presence and depth of ST segment depression. In principle, this measurement has a quite different meaning than exercise tolerance. Since ST segment depression is just one of the sequelae of myocardial ischemia, its presence and depth is partially related to the time relationship which it has relative to the development of symptoms. On the one hand, intolerable symptoms may develop before the onset of ST depression. On the other hand, ST segment depression may be profound before symptoms develop. In one study, the mean duration of exercise after the ST segment depressed 1 mm was 1.9 minutes with a range of 0-7 minutes. (78a) In another study, the depth of ST segment depression did not correlate with the extent of perfusion deficit by thallium scanning. (79) An important advantage of ST depression, however, is that it is a more specific finding than symptoms.

The depth of ST depression has been commonly thought to be an important prognostic variable. However, a much discussed article in the New England Journal of Medicine in 1981 (76) pointed out that this may not necessarily be the case. In

their series of 142 patients with exercise induced ST segment depression ≥ 2 mm who were followed for 5 years, the annual mortality was only 1.4%. Other studies do not have as dramatically low an annual mortality as the NEJM study, however these other studies support the idea that the depth of ST depression has a modest, but not profound, effect on prognosis. These studies are shown in Table 15. Patients were followed for 2-4 years.

Table 15. Medical Annual Mortality of Patients According to Severity of Exercise Induced ST Depression

Center / ST Depression	Annual Mortality (%)
Seattle (74)	
ST present	} no difference
ST absent	}
Boston University (75)	
> 2 mm ST	3
1-2 mm ST	2.5
0-1 mm ST	0.5
Duke (78)	
≥ 1 mm ST	6
< 1 mm ST	3
NHLI (72)	
≥ 2 mm ST	4
< 2 mm ST	2

Abbreviations: ST = Exercised induced ST depression

In the randomized European Cooperative Study of symptomatic patients with good ventricular function, patients were divided into those who had ≤ 1 mm ST depression and those who had ≥ 1.5 mm ST depression during exercise. After 5 years of followup, the patients with the least ST depression showed no difference in prognosis with surgery. However, the patients with the most ST depression showed a modest, but significant improvement in prognosis with surgery. (80) These results are shown in Figure 11.

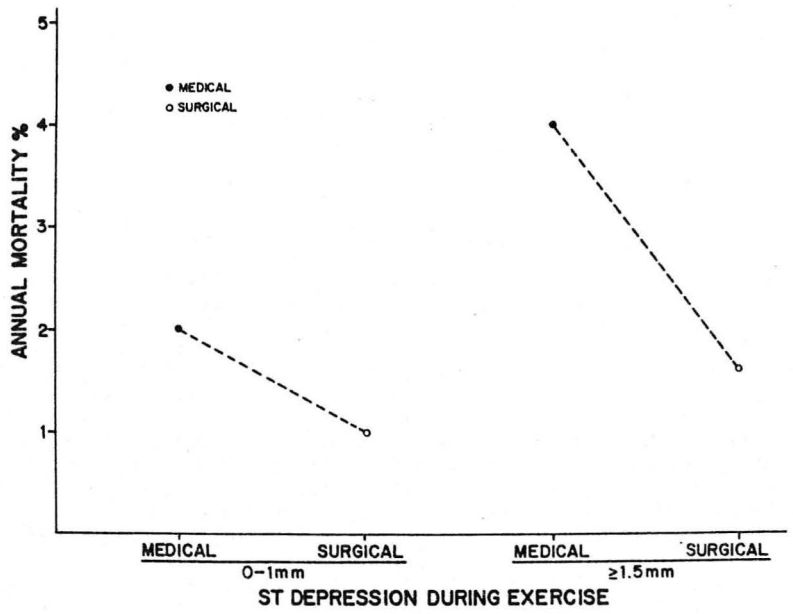

Figure 11. Medical and surgical annual mortality of patients subdivided according to depth of ST depression during exercise. Data is from randomized European Cooperative Study

Newer methods for assessing the reaction of coronary flow and ventricular function to stress using radionuclide techniques have recently been developed. (81-83) After time has elapsed to measure the mortality after these tests have been done, they may contribute to prediction of mortality.

The extent to which myocardium is in jeopardy should be assessed by a combination of symptoms and exercise test tolerance

When symptoms and the exercise test suggest that there is little myocardium in jeopardy, medical prognosis is very good

When symptoms and the exercise test suggest that much myocardium is in jeopardy, medical prognosis is not very good

When the exercise tolerance is poor, coronary anatomy is a strong independent predictor of prognosis

VA Study of Non Invasive Risk Factors

In the VA Cooperative Study of stable angina, patients were classified by a non invasive risk profile. While this risk profile does not fit neatly into the scheme of this discussion, the study does make several important points. The patients all had stable angina and had not had an infarct within the previous 6 months. Patients were assigned a risk based on the following 4 variables in decreasing order of importance; ST depression on resting EKG, history of infarction, history of hypertension, and class III-IV functional status. Patients were then divided into 3 equal sized groups based on the magnitude of this risk factor. Although just where a patient would fit into this classification scheme is difficult to conceptualize, patients in the low risk tercile had either none or only one of the risk factors except ST depression. Patients in the high risk tercile usually had 3 risk factors.

After being followed for 5 years, the medical annual mortality of the patients with combined 1, 2, and 3 vessel disease randomized to medical therapy is shown in the first column of Table 16. (10,14,15)

Table 16. Medical Annual Mortality of Patients Subdivided According to VA Risk Tercile and Coronary Anatomy

		Vessel Disease			
Risk Tercile	Combined 1, 2, 3	1	2	3	Left Main
Low	1	1	1.5	2	2
Mid	4	3	3	5	12
High	7	3	6	8	16

The dependence of the medical annual mortality on the risk tercile is apparent. From the other columns in Table 16 it is also apparent that the coronary anatomy

further predicts prognosis within each risk group. Notice the resemblance of this relationship to that between exercise tolerance and coronary anatomy. All coronary anatomy groups have a good prognosis in the low risk tercile. However, coronary anatomy is a strong predictor of prognosis in the high risk tercile.

When the patients in the VA Study who were randomized to surgery were subdivided into the same risk terciles as the medical patients, there was little difference in the surgical annual mortality between risk groups. (14,15) This is shown in Table 17.

Table 17. Medical and Surgical Annual Mortality of Patients According to Risk Tercile

	Annual Mortality (%)			
	No Left Main Disease		Left Main Disease	
Risk Tercile	Medical	Surgical	Medical	Surgical
Low	1	3	2	5
Mid	4	3	12	2
High	7	2	16	3

Therefore, it seems that surgical annual mortality is fixed at 2-3% regardless of coronary anatomy or non invasive risk tercile. This table is some evidence for the concept that CABG "fixes" survival to an annual mortality of about 2-3%, (or even better due to continuing improvement in surgical technique), regardless of the expected medical survival. The difference in medical and surgical annual mortality would, therefore, depend on the medical annual mortality.

Post Myocardial Infarct Period

Evaluation of the patient in the post-infarction period has recently been emphasized. This period encompasses the time from approximately 1-3 weeks post infarct to 6-12 months post infarct. Care of the patient before 1-3 weeks is generally considered under the topic of acute infarct care, while care of the patient after 6-12 months is generally considered under the topic of decreased ventricular function. The reason that the post infarct period is receiving so much attention is that mortality is high during this time period. Mortality is generally estimated at 10-15% (34,84,85,86,87) in the first year, although some estimates are lower. (88,89,90) If high risk patients can be detected, then treatment, especially by surgery, may improve the prognosis. Much more information is available on predicting medical prognosis, however, than is available on surgical prognosis. Another reason why evaluation during the post infarct period has been undertaken so widely is that this time period is both a convenient and psychologically appealing time to undergo evaluation.

The high mortality rate in the post infarct period can best be explained in one of two ways. The first explanation is that there is some unmeasurable factor present after an infarction that predisposes the patient to die. In general, this instability would seem to best be managed by a conservative plan of management. Such is probably the rationale behind the longstanding admonition to not perform non cardiac surgery on patients for 6 months after an infarct. The other explanation as to why patients have a high mortality in the post infarct period is that the infarct establishes a new pattern of coronary artery anatomy, ventricular function, and myocardial jeopardy. Patients who are at high risk of dying because of this new pattern would tend to die early. After elimination of the high risk patients, the mortality would decrease. This explanation would favor a more aggressive plan of management. Such is the logic behind the current interest in aggressive early evaluation of the patient after an infarct.

Initially, the safety of diagnostic techniques and CABG soon after infarction was of some concern. However, this fear is not justified. Maximal exercise tests (defined as an end point of symptoms) have been shown to be safe at 3 weeks post infarct, (34,88,90,78a) and submaximal tests (defined as an end point of symptoms, heart rate of 115-130, or Stage I of Bruce protocol, whichever comes first) have been shown to be safe at 11-19 days. (95,83,86,91,92) Since a submaximal test is performed to the same workload as a maximal test in the sickest patients, that is those patients who have symptoms before a heart rate of 120 or Stage I, it is probable that maximal tests are also safe at less than 3 weeks. However, this has not been documented. This point is of practical concern since many patients are discharged prior to 3 weeks post infarct. Cardiac catheterization has also been shown to be safe in the post infarct period. (93,88,84,89) These statements about the safety of exercise testing and cardiac catheterization are generalizations. As in the non post infarct period, judgement must be exercised in the selection of patients for these tests. CABG seems to carry about the same operative mortality in the post infarct period as at other times. (94) Earlier studies implied that operative mortality is higher in the post infarct period. (95) However, the indications for surgery in these studies were generally urgent ones for the complications of infarction.

Many variables have been studied in an attempt to predict the prognosis of a patient after an infarct. In general, the location of an infarct, whether it is transmural or subendocardial, and whether previous infarcts were present have been of little help in predicting prognosis. (84,85,96,96,86) Multivessel disease is found in about 50% of infarcts. Multivessel disease is found slightly more commonly in inferior infarcts than in anterior infarcts, however, the difference in incidence is small. (93,88,84,34,89) The variables which have been found to be most helpful in predicting prognosis after an infarct are the same ones which are helpful in the non post infarct period. Thus, the value of coronary anatomy, ventricular function, and the exercise test and symptoms in predicting the prognosis of a patient after an infarct will be reviewed.

Coronary Anatomy

The influence of coronary anatomy on the medical prognosis of patients followed 1-3 years in 4 studies is shown in Figure 12. (88,84,89,94) In these studies, the results are usually given at the end of the studied time period, and for this graph were reduced to annual mortality by dividing by the number of years followed. This

method assumes that the patients died evenly over the timer period followed. This assumption may falsely lower the annual mortality for the first year post infarct. Nevertheless, the important influence of coronary anatomy on survival is evident.

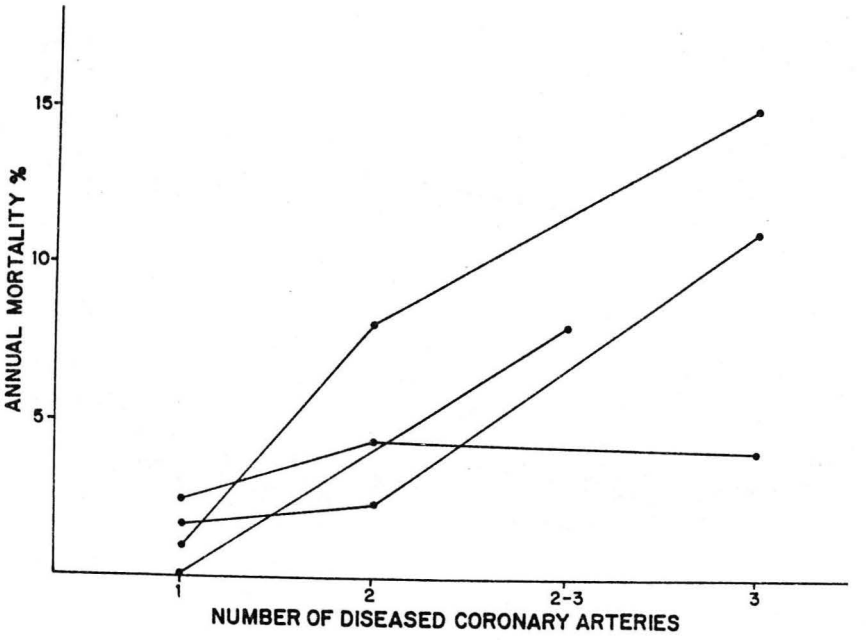

Figure 12. Medical annual mortality of post infarct patients subdivided according to coronary anatomy.

There is only 1 study in which medical and surgical prognosis was compared relative to coronary anatomy. (94) This is a retrospective study in which 43 patients had CABG performed an average of 99 days post infarct. These patients were compared to 51 patients who were surgical candidates, but not operated upon. Patients were followed 3 years. The overall annual mortality was 6% for patients followed medically and 3% for patients after surgery. The medical and surgical annual mortality relative to coronary anatomy for this same study is shown in Figure 13.

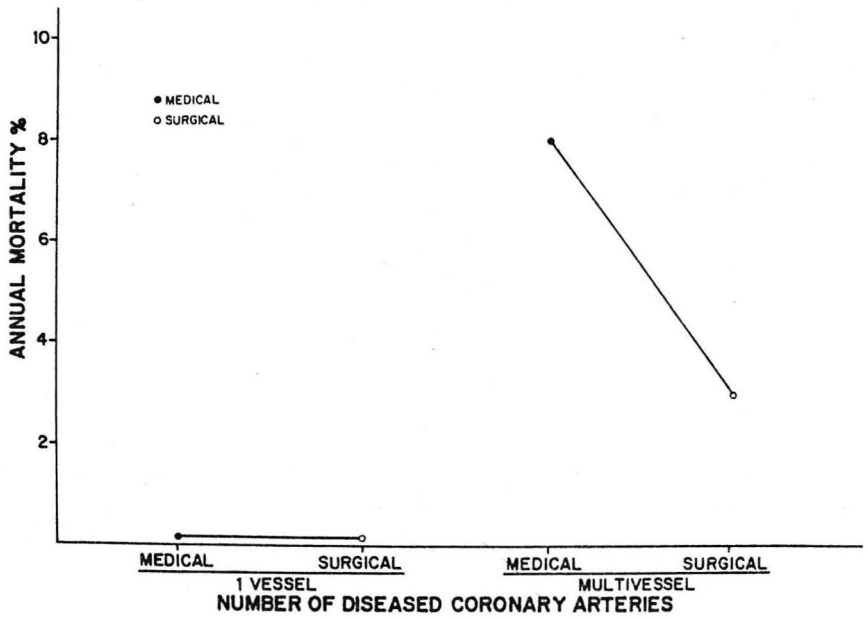

Figure 13. Medical and surgical annual mortality of post infarct patients subdivided according to coronary anatomy. The study was not randomized.

As in patients not in the post infarct period, the advantage of surgery for improving prognosis seems to be in the patient with multivessel disease.

Ventricular Function

Ventricular function is the strongest predictor of medical prognosis in the post infarct period of the variables that have been studied. The relationship of ejection fraction (normal > .50) to annual mortality in 8 studies is shown in Table 18. The followup periods in most studies are 1 year.

Table 18. Medical Annual Mortality of Post Infarct Patients Subdivided According to Left Ventricular Function

Center	Ejection Fraction	Annual Mortality (%)
Netherlands (88)	< .30	16
	> .30	1
Johns Hopkins (84)	< .30	34
	.30 - .39	7
	.40 - .49	4
	> .50	2
Australia (89)	< .50	7
	> .50	1
San Diego (98)	< .52	16
	> .52	0
Johns Hopkins (97)	< .40	31
	> .40	0
Alabama (94)	< .40	10
	> .40	4
New Zealand (87)	< .50	7
	> .50	0
NHLBI (92)	< .35	46
	> .35	0

The relationship of mortality to ventricular function shown on this table is very strong.

Although consideration of the relationship of ventricular ectopic activity to mortality is beyond the scope of this discussion, the interrelationship of ventricular ectopic activity and ventricular function should be pointed out. Approximately 90-93% of patients with complex ventricular ectopic activity have ejection fractions below .35 - .40. (97,92) It is unclear whether measurement of the degree of ventricular ectopic activity adds any prognostic predictive power over measurement of ventricular function alone. (99,97,92)

In the retrospective study comparing medical and surgical prognosis cited in the previous section on coronary anatomy, the results were also divided according to ventricular function. (94) These results are shown in Figure 14. Notice that annual mortality was lower with surgery with both good and poor ventricular function. However, also notice that both medical and surgical annual mortality is increased with poor ventricular function.

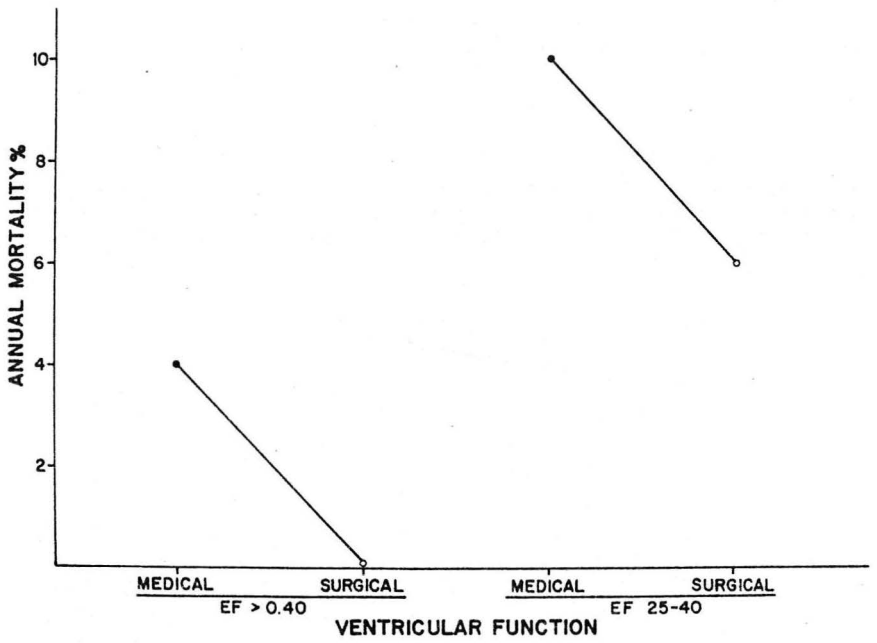

Figure 14. Medical and surgical annual mortality of post infarct patients subdivided according to ventricular function. The study was not randomized.

Symptomatic Status

Other than patients who have severe angina, the symptomatic status of patients in the immediate post infarct period is difficult to judge because of the limited activity during this period.

Patients who have angina at rest accompanied by EKG changes at less than 10 days after an infarct have a particularly bad prognosis. The mortality at about 6 months post infarct varies from 33% if the EKG changes are at the site of infarct to 72% if the EKG changes are at a site distant from the infarct (i.e. "ischemia at a distance"). (100)

At the other end of the spectrum are asymptomatic patients. In a randomized study, patients who were asymptomatic after having sustained at least 2 infarcts had a remarkably low annual mortality of only 2% after 5 years of followup, whether treated medically or surgically. (87) The unexpected result of this study, of course, was the good prognosis of patients treated medically. Partially accounting

for this good prognosis was the exclusion criteria for the study. Patients with angina, left main disease, or heart failure were excluded. In addition, the study did not not begin until 2 months after the infarct and surgery was not performed until an average of 5 months after the infarct.

Exercise Test

As in the non post infarct period, the workload that the patient can achieve before he has to stop for any symptom has prognostic value. (88,86) The dividing point between low and high workload in the 2 studies in Figure 15 are not the same, but the trend is apparent.

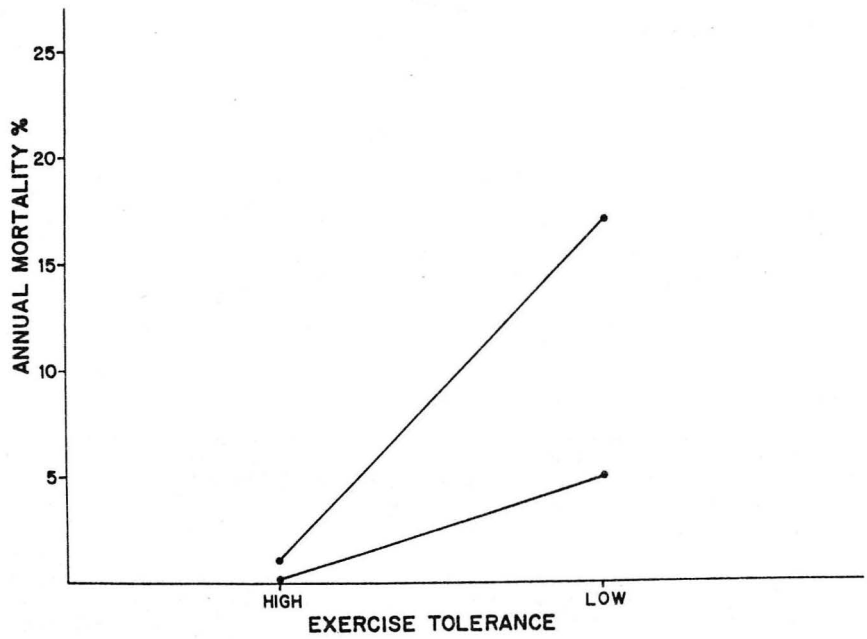

Figure 15. Medical annual mortality of post infarct patients subdivided according to amount of exercise tolerance.

The value of ST segment changes during the exercise test for predicting medical prognosis has been widely accepted since it was advocated in a review in the New England Journal of Medicine in 1982. (34) While ST segment changes are of value for predicting prognosis, it should be realized that most of the dramatic evidence for the NEJM review came from one publication. (85) The evidence from 3 other publications are not as dramatic. (88,86,91) The results from these 4 studies are shown in Figure 16.

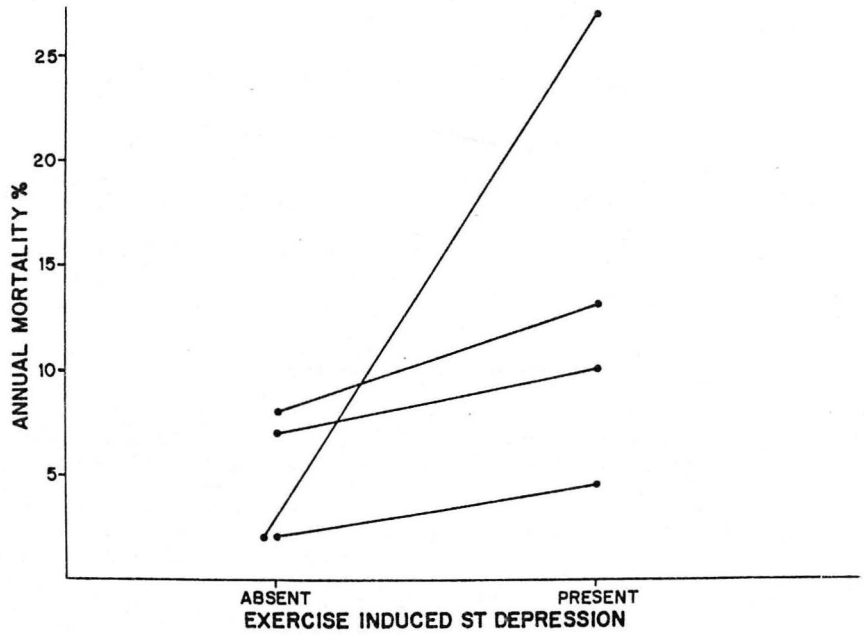

Figure 16. Medical annual mortality of post infarct patients subdivided according to the presence or absence of ST depression during exercise.

The presence or absence of pain during the exercise test seems to be of little prognostic value. (85,86)

Several other methods of judging the amount of jeopardized myocardium and hence estimating prognosis deserve mention. The use of radionuclide angiography to measure the ejection fraction and its change with exercise is being actively investigated at this institution. (82,83) The present results suggest that it

is valuable in predicting cardiac events in the post infarct period. However, other investigators are guarded in their enthusiasm for radionuclide angiography during exercise for predicting post infarct mortality. (92) Pulmonary edema at the time of infarct, even when the ejection fraction measured later is good (> .45) is regarded by some as a sort of stress test for jeopardized myocardium. In one study, patients who recovered from pulmonary edema and had a good ejection fraction had a very high mortality, (101) (approximately 40% at one year although figures not exact). Finally, the catheterization assessment of myocardium in jeopardy, as judged by contractile myocardial segments fed by critically diseased vessels, has prognostic value. (94,84) In one study, (84) patients with jeopardized segments had a 15% mortality while patients without jeopardized segments had a 0% mortality.

In the post infarct period:

Ventricular function is the best predictor of medical prognosis

Coronary anatomy, exercise tolerance, and ST segment depression on exercise testing have prognostic value, but less than ventricular function

Little information is available on the value of surgery for improving prognosis

Summary and Conclusions

I have approached this discussion from a physiologic point of view. I will conclude by briefly summarizing the discussion from a more clinical point of view. This conclusion will consider only the diagnostic and therapeutic steps concerned with predicting or improving prognosis. In many clinical instances, some studies will have already been performed to clarify diagnoses. In other instances, surgery will be performed to relieve symptoms.

From a prognostic standpoint, the diagnostic approach can be aggressive in which case all patients have an exercise tolerance test and cardiac catheterization. Alternatively, a more conservative step wise approach can be followed. In Figure 17, if catheterization is performed at the outset, the diagnostic options can be passed over. In the case of post infarction patients, the evaluation should be done just prior to discharge, or at least no later than 3 weeks after the infarct. Obviously, patients who are unstable or in clinical heart failure should have an individualized approach and not necessarily the approach outlined on the diagram.

A diagram of the suggested approach is shown below:

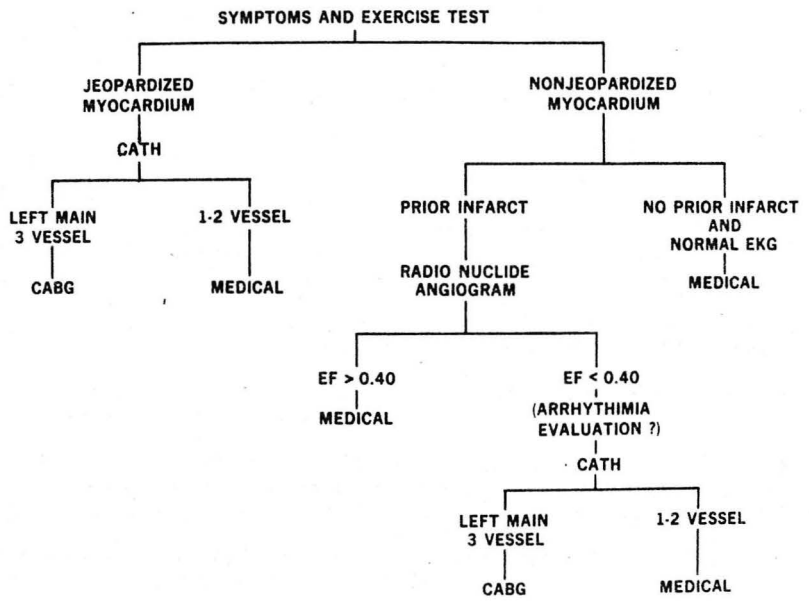

Figure 17. A suggested basic approach to the diagnosis and treatment of patients with coronary disease. The emphasis is on predicting and possibly improving prognosis.

This diagram suggests a straightforward approach which is supported by the previous discussion. However, the recommendations for the patient with an ejection fraction < .40 are based on conjecture. Certainly CABG should only be performed if myocardium appears to be in jeopardy as assessed functionally by symptoms and exercise testing, or anatomically by coronary angiography. However, lethal arrhythmias, secondary to the scarred myocardium, are an important possibility. These arrhythmias will not be cured by CABG. (27-29) The best way to approach these arrhythmias or potential arrhythmias is as yet uncertain. The two most common diagnostic approaches are either prolonged monitoring (102) or ventricular stimulation. (30-33) If potentially lethal arrhythmias are found, they can possibly be treated by drugs, surgery or an implantable defibrillator. (33)

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